standard measurements for GFR and to the small sample size of these studies. Therefore one should rather stress the need for the construction of a proper cystatin C based GFR equation relying on a large patient cohort as it was the case for the creatinine based MDRD GFR equation.3

The formula we chose for GFR calculation from cystatin C was the indeed based PETIA serum cystatin C assay.4 However it was the only study dealing with a relatively large sample of adult patients with a high proportion of healthy persons and we expected that this casemix would be more comparable to ours. On the contrary, the equation proposed by Lambermont et al. refers to a casemix of patients with known chronic kidney disease (CKD), which was not the case for the majority of our patients.

We nevertheless performed the logistic and the Cox models with 10 published equations2 and found results similar to those published in our manuscript.5 We found that, using the method described in our manuscript for the multivariable analysis, the models selected the EuroSCORE and the GFR estimated from serum Cystatin C with similar odds ratio, confidence intervals and P-values and this regardless the formula used for the GFR estimation. It appears that the results we observed are not particularly sensitive to which formula is used for the estimation of the GFR from serum cystatin C. The conclusions of our work remain therefore valid.

References

Table 1 GFR estimated from 10 cystatin based equations2

<table>
<thead>
<tr>
<th>Cyst C (mg/L)</th>
<th>PENIA</th>
<th>PETIA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Le Bricon (n = 25, renal transplant)</td>
<td>Hoek (n = 123, CKD)</td>
</tr>
<tr>
<td>0.8</td>
<td>101.5</td>
<td>96.1</td>
</tr>
<tr>
<td>1</td>
<td>82.0</td>
<td>76.0</td>
</tr>
<tr>
<td>1.1</td>
<td>74.9</td>
<td>68.7</td>
</tr>
<tr>
<td>1.5</td>
<td>56.0</td>
<td>49.2</td>
</tr>
<tr>
<td>2</td>
<td>43.0</td>
<td>35.9</td>
</tr>
<tr>
<td>4</td>
<td>23.5</td>
<td>15.8</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Tan (n = 40, adults)</th>
<th>Larsson (n = 100, adults)</th>
<th>Grubb (n = 451, adults)</th>
<th>Grubb#* (n = 451, adults)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>102.0</td>
<td>141.6</td>
<td>145.4</td>
<td>126.0</td>
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<tr>
<td>0.8</td>
<td>80.2</td>
<td>99.4</td>
<td>99.2</td>
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<tr>
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<td>72.3</td>
<td>85.5</td>
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<tr>
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<td>52.3</td>
<td>49.5</td>
<td>43.7</td>
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<tr>
<td>2</td>
<td>14.9</td>
<td>11.1</td>
<td>9.2</td>
<td>8.4</td>
</tr>
</tbody>
</table>

Cyst C, serum cystatin C; CKD, chronic kidney disease; PENIA, particle enhanced nephelometric immunoassay; PETIA, particle enhanced turbidimetric immunoassay.

*Grubb equation using the gender variable. Calculations were made for male gender.

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Long-term follow-up of primary prophylactic implantable cardioverter-defibrillator therapy in Brugada syndrome

In reference to the original and interesting study by Sarkozy et al.,1 we would like to point out the following comments regarding their inclusion criteria in this original study.

The study found the difference in appearance of coved-type ECG during follow-up of patients with baseline type II, type III, and normal ECG, respectively (56, 17, and 0%), and normal ECG, respectively (56, 17, and 0%). Although type II and III are not diagnostic, these findings indicate that they do not have the same significance. Moreover, normal resting ECG in the so-called high-risk patients seems not to have dynamic changes, leading to diagnostic coved type. Thus, it is not clearly mentioned whether each patient with baseline normal resting ECG (nine patients) has the following risk factors: positive family history of sudden death (55% of all 47 studied), syncope prior to ICD (55%), both (30%), or inducible EP study (83%); considering that spontaneous coved type had not been seen during the follow-up period. Did they find SCNSA mutation or other mutation in these nine

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patients with normal resting ECG? What is the age distribution in the follow-up period? Knowing that if they were too young, BS phenotype might not have enough time to appear. None of them had appropriate shock. ICD implantation in patients with non-spontaneous coved-type pattern in conjunction with family history of sudden death and positive EP study is considered as class IIb. The consensus did not mention specifically the place of normal spontaneous ECG in the assessment of subjects with other high-risk factors of sudden death in Brugada syndrome. However, the authors’ findings concerning baseline normal resting ECG seem to be confident with the highest negative predictive value as marker of sudden death. The authors’ conclusion did not notice this fact, which is relevant from our point of view.

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Long-term follow-up of primary prophylactic implantable cardioverter-defibrillator therapy in Brugada syndrome: reply

We are thankful for the excellent remarks of Dr Bonny regarding the different frequency of spontaneous type I ECG during follow-up among patients with baseline type II, type III, and normal ECGs. First, we would like to answer your question regarding the indication for ICD implantation in the nine patients with normal baseline ECG. Seven of the nine patients had undocumented syncope, which is in itself an indication for an ICD implantation. These seven patients were all adults, two of them also had atrial fibrillation and four of them were inducible to VF during an EP study. The eighth patient was a 6-year-old asymptomatic child identified during family screening of a large family with Brugada syndrome, who developed ventricular fibrillation during the class I AAD test following half dose of the ajmaline and a strongly positive response. This patient was also a carrier of the SCNSA mutation identified in her family. She was the only patient who had an SCNSA mutation identified. The ninth patient was asymptomatic, but had a strongly positive family history of sudden death (three first-degree relatives dying suddenly at a young age) and was inducible to VF during the EPS.

The reason why the importance of the presence of spontaneous type I ECG at follow-up. Conversely, in patients in whom baseline only normal ECGs are documented and have no type I ECG during follow-up, every fifth ECG is type II or III. In summary, we completely agree with you that the presence of spontaneous type I ECG is a marker of increased risk as it has also been observed in previous studies. However, we believe that, given the extreme variability of the ECG in this syndrome and the small patient numbers in our study, further studies are necessary to make meaningful conclusion about the predictive value of baseline type II, III, and normal ECGs.

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